

Biology and control of *Rhizoctonia solani* on rapeseed : A Review

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Résumé de l'article

Le *Rhizoctonia solani* AG2-1 est le principal agent pathogène causant la fonte des semis chez les plantules et les plantes matures (dessèchement prématuré des pieds) sur le colza et le canola (*Brassica napus* et *B. râpa*) dans l'Ouest canadien et aux États-Unis; les isolats du groupe AG4 affectent surtout les plantes adultes en causant le rhizoetone commun. L'infection des plantules par AG2-1 est favorisée par les températures fraîches au moment du semis, alors que les températures chaudes survenant tard au cours de la saison de croissance favorisent davantage l'infection des plantes matures par les isolats de AG4. Des données d'enquête montrent que le développement de la maladie est favorisé par une humidité du sol élevée, de bas niveaux d'azote, de phosphore et de potassium, ainsi que par des teneurs élevées en cuivre dans les sols à texture fine. Une résistance modérée chez la moutarde (*Sinapis alba*) et quelques autres espèces semblables semble être d'origine génétique et elle devrait être utilisée dans des programmes d'amélioration génétique. La carboxine et l'iprodione en mélange avec des insecticides gamma-HCH sont homologués au Canada comme traitements de semence afin de réprimer la fonte des semis et la pourriture racinaire des plantules, mais ce traitement ne réprime pas le dessèchement prématuré des pieds.

Biology and control of *Rhizoctonia solani* on rapeseed : A review

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Rhizoctonia solani AG2-1 is the principal pathogen causing damping-off and seedling and mature plant root rot (brown girdling root rot) in oilseed rape and canola (*Brassica napus* and *B. rapa*) in western Canada and the United States; AG4 isolates mainly attack adult plants and cause basal stem rot. Seedling infection by AG2-1 is favoured by cool weather at the time of planting, whereas warm weather late in the growing season is more conducive for infection of mature plants by AG4 isolates. Survey data show that disease development is favoured by high soil moisture, low levels of nitrogen, phosphorus and potassium and high levels of copper in fine-textured soils. Moderate resistance in condiment mustard (*Sinapis alba*) and some other species appears to be genetically controlled and should be utilised in breeding programmes. Carboxin and iprodione in mixtures with insecticide gamma-HCH are recommended in Canada as seed treatments to control damping-off and seedling root rot, but do not control brown girdling root rot.

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INTRODUCTION

The oilseed *Brassica* species [*B. napus* L., *B. rapa* L., and *B. juncea* (L.) Czern. and Coss.] and condiment mustard (*Sinapis alba* L.), represent annual revenues of over a billion dollars to the Canadian economy. An important disease of oilseed rape and canola (*B. napus* and *B. rapa*) caused by *Rhizoctonia solani* Kühn is distributed worldwide, and poses an economic threat to canola crops in the prairie regions of western Canada (Ellis 1983; Kataria and Verma 1992). The population of *R. solani* that infects oilseed rape and canola is mainly composed of anastomosis groups (AGs) AG2-1 and AG4 which cause pre- and post-emergence damping-off, seedling root rot and basal stem or foot rot (brown girdling root rot) of adult plants (Gugel *et al.* 1987; Kaminski and Verma 1985; Yitbarek *et al.* 1987).

THE DISEASES

Pre- and post-emergence damping-off, and root rot of seedlings and adult plants are serious in oilseed rape in western Canada (Berkenkamp 1972; Berkenkamp and Vaartnou 1972; Ellis 1983; Rimmer and Platford 1982; Sippell *et al.* 1985b). Although disease severity varies with region and weather at planting time, a majority of rapeseed crops in western Canada are affected to some extent each year. Root rot incidence has generally been low in Manitoba and Saskatchewan (Petrie and Vanterpool 1970; Rimmer and Platford 1982), but serious in Alberta. Incidence of 80-100% with major losses of plant stands were recorded in the Peace River region (lat. 55-57°N, long. 117-120°W) of Alberta (Ellis 1983; Sippell *et al.* 1985a,b). The predominant type of root rot in the Peace River region is called brown girdling root rot due to the production of large, sunken light brown lesions on the tap root and lateral roots. Girdled plants are left with only a short stub of the tap root and are vulnerable to desiccation and lodging. Brown girdling root rot commonly results in poor pod development, pod sterility, shrivelled seed, low seed weight, lodging and uneven ripening of plants. Annual yield

losses of 30% were attributed to this disease in surveys conducted in the Peace River region in 1983 and 1984 (Sippell *et al.* 1985a).

THE CAUSAL AGENTS

Rhizoctonia solani Kühn [teleomorph: *Thanatephorus cucumeris* (A.B. Frank) Donk] infects many members of the Brassicaceae (Akhtar 1969; Benson 1973; Chauhan and Saxena 1974; Flentje *et al.* 1963; Gupta 1985; Henis *et al.* 1978; Homma and Ishii 1984; Huber *et al.* 1992; Johnson *et al.* 1992; Kataria and Verma 1992; Keyworth and Dow 1960; Mahmood and Iqbal 1982; Verma and Kataria 1990; Weber 1987; Williams and Walker 1966). *R. solani* is the principal causal agent of damping-off and seedling and adult plant root rot of rapeseed in western Canada, but *Fusarium* spp. and *Pythium* spp. also are frequently associated with root rot in that region (Acharya *et al.* 1984; Berkenkamp and Degenhardt 1974; Calman and Tewari 1987; Calman *et al.* 1986; Gugel *et al.* 1987; Kaminski and Verma 1985; Petrie 1973; Petrie and Vanterpool 1966, 1970; Sippell *et al.* 1985b; Vanterpool 1960, 1963, 1974; Yitbarek *et al.* 1987). In Finland, damping-off due to *R. solani* occurred in 90% of spring rape (*B. napus* and *B. rapa*) fields surveyed during 1981-82 (Tahvonen *et al.* 1984). Several brassicaceous weeds, including ball mustard [*Neslia paniculata* (L.) Desv.], stinkweed (*Thlaspi arvense* L.) and shepherd's purse [*Capsella bursa-pastoris* (L.) Medic.], also are affected by the brown root rot caused by *R. solani*. Besides root and collar rot, *R. solani* also causes a severe leaf blight of oilseed rape and mustard in India. Diseased leaves develop brownish patches with chlorotic margins and abscise prematurely (Chauhan and Saxena 1974; Gupta 1985). The leaf blight phase of *R. solani* has not been reported from temperate countries.

THE PATHOGEN

Characterization of *R. solani* populations

Isolates of *R. solani* from field soils and diseased seedlings and adult plants of

rapeseed in western Canada have been characterized into anastomosis groups (AGs) by pairing (hyphal fusion) with known tester isolates of the pathogen. Among the 83 isolates of *R. solani* obtained from rapeseed plants in Saskatchewan, 48% were assigned to AG2-1, and 50% to AG4, and the remaining 2% did not anastomose with any AG tester (Kaminski and Verma 1985). Among 69 isolates from rapeseed in west central Alberta, 41% belonged to AG2-2, 32% to AG2-1, and 14% to AG4; the remaining 13% were not assignable to any AG tester (Hwang *et al.* 1986). Gugel *et al.* (1987) found that all 68 *R. solani* isolates obtained from rapeseed plants in the Peace River region of Alberta belonged to AG2-1, but those from soils of that region were 59% AG2-1, 25% AG4, and 16% other AGs. Populations of *R. solani* in soil of Saskatchewan rapeseed fields belonged to AG2-1 (36%), AG2-2 (6%), AG3 (4%), AG4 (53%), and AG5 (2%) (Yitbarek *et al.* 1987). The AG4 isolates were detected in soils of all textures, but AG2-1 isolates were confined to loam, silty clay loam, and silty clay soils. In these surveys, isolates from field-infected seedlings belonged to AG2-1 and AG4, with frequencies of 94 and 6%, respectively. Among isolates from adult plants, 69% were AG2-1, 29% were AG4 and 2% were AG2-2 (Yitbarek 1987; Yitbarek *et al.* 1987). All *R. solani* isolates from rapeseed in Indiana (U.S.A.) belonged to AG2-1 (Huber *et al.* 1992).

Cultural characteristics

The isolates of *R. solani* from diseased rapeseed plants varied in growth rate, hyphal coloration and sclerotial formation. Colonies of AG2-1 on potato dextrose agar (PDA) were light brown to brown, with concentric zones of appressed and aerial mycelium. Sclerotia were produced in various sizes and numbers, and were distributed in the zones of aerial mycelia. The AG2-2 isolates had sparse, brown appressed mycelia. Colonies of AG4 on PDA were almost white at first, but later turned brown-grey with a leathery, appressed or mealy textured mycelium, and formed few, minute, light brown sclerotia that were embedded in the agar, mostly around the inoculum disc. Mean growth rates of AG2-1 and

AG4 isolates from rapeseed were 0.34 and 0.51 mm h⁻¹, respectively, at 18°C (Hwang *et al.* 1986; Kaminski and Verma 1985).

Growth rates of isolates within AG2-1 and AG4 differed slightly but significantly and differences between the two AGs were large (Kaminski and Verma 1985). Isolates of AG2-1 grew at 2°C but not at 36°C and optimally at 24°C, whereas AG4 isolates did not grow at 2°C, grew slowly at 36°C and optimally at 26°C. Temperatures in the range of 18-30°C supported luxuriant growth of both AGs, and AG4 isolates had a higher growth rate than AG2-1 isolates at these temperatures.

Virulence of *R. solani* isolates

Virulence of isolates differed considerably and was influenced by their source. Isolates of AG2-1 from seedlings and adult rapeseed plants were generally more virulent than those of AG2-2 and AG4, but virulence differed markedly among isolates of the same AG (Gugel *et al.* 1987; Kaminski and Verma 1985; Yitbarek 1987; Yitbarek *et al.* 1987). Isolates from seedlings and adult plants were more virulent than those of the same AG from soil. Isolates of AG2-1 and AG4 from seedlings were more virulent than isolates from adult plants. In tests on agar media, soil isolates induced less severe disease in hypocotyls and radicles than did isolates from seedlings and adult plants, and seedling isolates prevented seed germination more than the isolates from adult plants (Yitbarek 1987; Yitbarek *et al.* 1987). Soil isolates of *R. solani* AG9 were reported as mildly pathogenic on canola seedlings (Yang *et al.* 1996).

AG2-1 isolates are the chief causal agents of pre- and post-emergence damping-off and seedling root rot in rapeseed production areas of western Canada, and in the state of Indiana in the United States (Huber *et al.* 1992; Yitbarek *et al.* 1987). AG4 isolates mainly attack adult plants and cause basal stem rot late in the growing season. AG2-2 isolates, although pathogenic on rapeseed seedlings, are not as prevalent and wide-spread as AG2-1 and AG4 isolates in rapeseed fields (Gugel *et al.* 1987; Hwang *et al.* 1986; Kaminski and Verma 1985; Yitbarek *et al.* 1987).

Infection process

The sequence of events in the infection process was found to be similar in susceptible and resistant hosts (Yang *et al.* 1992a). Infection cushions were formed on hypocotyls of both susceptible and resistant hosts, but were more frequent and formed earlier on susceptibles. In hypocotyls of the susceptible cv. Westar, hyphal growth and infection cushions were observed within 24 h after inoculation, and parenchyma cell maceration was found within 2 d. In contrast, in resistant cv. Arda, infection cushions did not occur until 48-72 h, and cell maceration required 3 d. Infection was more rapid and deeper in susceptible than in resistant hosts. In all tissues of 'Arda', hyphae grew more slowly, branched less frequently, oriented irregularly, and were confined largely to the parenchymatous tissue of the cortex. Hyphae were not observed in endodermis or vascular tissues 5 d after inoculation (Yang 1989; Yang *et al.* 1992b). The formation of infection cushions on susceptible cv. Westar was similar to that on radish (*Raphanus sativus* L.) in which the hyphae grew parallel to the longitudinal axis of the hypocotyl, but on resistant cv. Arda they were similar to that on bean (*Phaseolus vulgaris* L.) in which hyphal growth was less regular (Dodman *et al.* 1968).

Factors affecting disease development

Incidence and severity of pre- and post-emergence damping-off and root rot in rapeseed fields is influenced by weather and soil conditions and by inoculum density of the anastomosis groups of *R. solani* in the soil. Seedling infection by AG2-1 isolates is favoured by cool weather after planting, whereas warm weather favours infection of mature plants by AG4 isolates (Teo *et al.* 1988).

Night/day air temperatures, isolate, inoculum density, and all possible interactions among these factors significantly affect incidence of pre-emergence damping-off in rapeseed grown in soilless potting mix (Yitbarek 1987; Yitbarek *et al.* 1988). In general, incidence of damping-off caused by AG2-1 and AG4 isolates increased progressively with increased inoculum density, but virulence of both isolates was affected principally by tem-

perature. At all inoculum densities tested (1400-22 800 viable propagules L⁻¹ soilless potting mix) in the 7:8°C and 7:12°C regimes, and at the lower inoculum densities in the 7:18°C regime the AG2-1 isolate caused more damping-off than the isolate of AG4. In contrast, the isolate of AG4 caused more damping-off than the isolate of AG2-1 at all inoculum densities in the 26:35°C regime, and at the lower inoculum densities in the 12:18°C and 19:25°C regimes. The two isolates caused a similar incidence of damping-off at the higher inoculum densities in the 7:18°C, 12:18°C, and 19:25°C regimes.

Effects of soil temperature on virulence of AG2-1 and AG4 isolates were found also in field experiments. When rapeseed was planted early before the soil warmed to 15°C, AG2-1 caused more damping-off than did AG4, while damping-off due to AG4 increased as the soil warmed to 20°C (Teo *et al.* 1988; Yitbarek 1987). The influence of soil temperature on virulence was also reflected in the frequency with which these two AGs were isolated from diseased seedlings and mature plants (Yitbarek *et al.* 1987). Isolates from seedlings were almost all AG2-1, whereas AG4 were isolated almost five times more frequently from mature plants than from seedlings.

Effects of temperature on *R. solani* isolates in rapeseed were similar to those for radish in which pre-emergence damping-off was more severe at 26°C than at 15, 20, 22, or 30°C, and 6000-10 000 propagules kg⁻¹ soil were required to induce 20-80% disease incidence at 15°C, compared with only 500-1500 propagules kg⁻¹ soil at 26°C (Benson and Baker 1974). In Japanese radish (*R. sativus*) AG2-1 was isolated most frequently from plants grown in winter; plants grown at other times of the year when conditions were warmer yielded more AG4 isolates. AG2-1 and AG4 isolates also showed maximum virulence at low and high temperatures, respectively (Homma and Ishii 1984).

Results of field tests in Saskatchewan showed no consistent effect of soil moisture on AG2-1 or AG4-induced pre-emergence damping-off in rapeseed (Teo *et al.* 1988; Yitbarek 1987). In the Peace River region of Alberta, however, surveys of brown girdling root rot from 1983

through 1985 showed that disease development was favoured by high rainfall and high soil moisture (Sippell *et al.* 1985a). Disease incidence and severity were highest in 1983, intermediate in 1984, and lowest in 1985, which coincided with declining annual rainfall from 318 mm in 1983, to 288 mm in 1984, and 161 mm in 1985. These surveys also recorded higher root rot severity in fields with low levels of nitrogen, phosphorus and potassium, and in fine-textured soils with high levels of copper.

CONTROL MEASURES

Plant resistance

Screening plant lines for resistance

All rapeseed or canola cultivars currently grown in Canada are susceptible to *R. solani* (Acharya *et al.* 1984; Yang 1989; Yang and Verma 1992). Using a mixture of 35 isolates of *R. solani*, not characterized into anastomosis groups, Acharya *et al.* (1984) evaluated 300 *B. napus* and *B. rapa* genotypes in a growth chamber, and found 10 lines, five from each of the two species, that were significantly more resistant to *R. solani* than commercial cultivars. All 10 lines were later found to be highly susceptible to a rapeseed isolate of AG2-1 (Yang 1989). Harrison (1987) evaluated over 200 accessions of *B. rapa* and *B. napus* in a naturally-infested field nursery and found significant differences in disease severity among the accessions.

In order to identify resistant genotypes within the oilseed brassicas and their close allies, growth chamber studies were initiated to screen germplasm against an isolate of *R. solani* AG2-1. Results of a test involving 16 cultivars of six *Brassica* species showed that pre-emergence damping-off was most severe in *B. carinata* Braun, *B. rapa*, *B. oleracea* L. and *B. napus*, and least severe in *B. juncea* and *B. nigra* (L.) Koch; lower pre-emergence damping-off in *B. juncea* and *B. nigra* may be related to faster emergence of seedlings in these than the other four species. Although all six species suffered severe post-emergence seedling root rot, damage was greater in *B. juncea* and *B.*

nigra. Significant differences in the levels of susceptibility/resistance to pre- and post emergence damping-off occurred among cultivars within the *Brassica* species (Kataria *et al.* 1993).

In another growth chamber study none of 122 cultivars/lines of *B. rapa*, *B. napus*, *B. juncea*, *B. nigra*, *B. tournefortii* Gouan, *B. carinata*, *B. oleracea* var. *capitata* L., *B. oleracea* var. *botrytis* L., *S. alba*, *R. sativus* and *Camelina sativa* L. evaluated were immune to AG2-1 although differences were found in susceptibility among and within genera and species (Yang and Verma 1992). Among 11 species tested, all eight lines of *S. alba* and one line of *C. sativa* consistently exhibited elevated resistance to *R. solani*, indicating that moderate resistance was genetically controlled and could be utilized in breeding programmes. Although interspecific crosses between *S. alba* and the *Brassica* spp. oilseed crops are very difficult to obtain, it may be possible to transfer the resistance to *R. solani* from *S. alba* to other commercial *Brassica* species. Successful transfer of beet cyst nematode resistance from *S. alba* to *B. napus* (Van Lelivelt *et al.* 1989), and interspecific hybridization between *B. juncea* and *S. alba* (Mohapatra and Bajaj 1987), and between *B. napus* and *S. alba* (Ripley and Arnison 1990) by means of embryo rescue strengthen this possibility.

Effect of host age

Rapeseed plants can be infected by isolates of *R. solani* AG2-1 and AG4 at all growth stages but acquire some resistance with age. Percent disease ratings of nine rapeseed and mustard cultivars inoculated at 0, 3 and 5 wk after planting showed that the older the plant at the time of inoculation, the lower the disease ratings; differences in disease ratings between 3 and 5 wk were highly significant (Yang and Verma 1992). Gugel *et al.* (1987) also studied effects of age of host at inoculation, anastomosis group (AG2-1 and AG4), and inoculum level (1, 2, or 3 *R. solani* infested rapeseed seeds plant⁻¹). Increasing plant age significantly declined percent disease severity for both AGs of *R. solani*, especially AG2-1. Also increasing inoculum level from 1 to 3 infested seeds plant⁻¹ significantly increased percent disease ratings.

Effect of bean endochitinase gene in transgenic rapeseed

Rapeseed plants containing high constitutive levels of bean endochitinase are more resistant to infection by *R. solani*, than are plants that lack the chimeric chitinase gene (Broglie *et al.* 1991). The enzyme endochitinase inhibited *R. solani* growth *in vitro* and induced morphological changes in the mycelia including swelling and disruption of hyphal tips.

In transgenic plants, *R. solani* penetrated the host cuticle and epidermis, and colonization, while normally restricted to the cortex, sometimes occurred in the xylem vessels. Vacuolization and lysis of hyphal cells, and reduced fungal biomass were common in transgenic plants, and correlated with chitin degradation (Benhamou *et al.* 1993). Constitutive expression of the bean endochitinase gene was considered partly responsible for observed protection against *R. solani* (Benhamou *et al.* 1993).

Transgenic canola cv. Westar containing the endochitinase gene (Broglie *et al.* 1991) did not control pre- and post-emergence damping-off and seedling and adult plant root rot induced by AG2-1 in the growth chamber or in the field (Verma, unpublished data). Transgenic canola may require both chitinase and glucanase to be resistant against any fungal pathogens including *R. solani*.

Antimicrobial compounds and phytoalexins

Camelina sativa is moderately resistant to *R. solani* AG2-1 (Yang 1989; Yang and Verma 1992), possibly due to the presence of antimicrobial compounds in the roots (Conn *et al.* 1994). Four antimicrobial compounds, capable of inhibiting mycelial growth of *R. solani* isolates were extracted and purified from *C. sativa* roots. Of the four compounds, 10-methylsulfinyldecylisothiocyanate and methyl 1-methylindole-3-carboxylate were produced in *C. sativa* roots only, while the phytoalexins, camalexin and methoxycamalexin were produced in roots, stems, leaves and siliquae (Conn *et al.* 1987, 1994).

Role of the plant cuticle

Cuticle penetration by *R. solani* is probably due to mechanical force created by

the growth of infection cushions (Bate-man 1970). A thicker cuticle increases resistance to this mechanical force, reduces frequency and delays penetration by the pathogen, and thus allows more time for mobilization of host defenses.

Brassica napus and *S. alba* plants acquire some resistance to *R. solani* AG2-1 with age (Gugel *et al.* 1987; Yang and Verma 1992). In a study determining differences in cuticle thickness and morphology of epicuticular wax, (Yang *et al.* 1992a) found that 1-wk-old seedlings of susceptible *B. napus* showed no obvious cuticle layer, but at 3 wk a cuticle was present with a concomitant increase in resistance to *R. solani*. Resistant 3-wk-old *S. alba* seedlings had much less disease and a significantly thicker cuticle layer than *B. napus* plants of the same age. These results suggest that the cuticle plays an important role in the resistance of *S. alba* and older plants of *B. napus* to infection by *R. solani*. Similar results have been reported in bean plants (Stockwell and Hanchey 1983).

Hypocotyl surfaces from 3-wk-old plants of susceptible *B. napus* contained more amorphous layer of two types of wax crystals than did those of resistant *S. alba* (Yang *et al.* 1992a). Fewer wax deposits on *S. alba*, a resistant species, suggests that wax may not be important in affording resistance to *R. solani* hypocotyl rot at the seedling stage. This is not in agreement with observation in rice in which a positive correlation between resistance to *R. solani* and wax concentration was reported (Massaquoi and Rush 1987).

Host calcium content and resistance

Calcification of host cell walls was reported to be a mechanism of resistance to *R. solani* in some crops (Vidhyasekaran 1988). Besides affecting pectin metabolism and cell membrane permeability in the host, calcification decreases the availability of certain micronutrients required by pathogens. In bean, lesion delimitation and resistance of older plants resulted from increased calcium pectate accumulation in and around developing lesions, because polygalacturonase produced by the pathogen was incapable of

hydrolysing calcium pectate (Bateman 1964). He considered that *R. solani* was not able to destroy all calcium pectate in beans by producing oxalic acid. However, enhanced resistance in older crucifer plants was not associated with the increased calcium content of the host. More calcium oxalate crystals were found on hypocotyls of the susceptible *B. napus* cv. Westar than on hypocotyls of resistant *S. alba* cv. Arda, and *C. sativa*; 1-wk-old plants had more calcium oxalate than did 3-wk-old plants (Yang *et al.* 1993). Total calcium content did not differ in non-infected hypocotyl tissues of the three plant genera, and did not increase with increasing age of plant tissue. Production of oxalic acid in culture and the formation of calcium oxalate crystals in infected tissues suggests that calcium in plant cells is sequestered by oxalic acid produced by *R. solani*. However, calcium content of the host is not associated with resistance in *S. alba* and *C. sativa*. Factors, including phytoalexins that inhibit growth of the pathogen and prevent oxalic acid production may have a greater role in generic and age-related resistance. Crucifers resistant to *R. solani* produce larger amounts and different kinds of phytoalexins and antimicrobial compounds than do susceptible crucifers (Conn *et al.* 1987, 1994; Tewari *et al.* 1988).

Chemical control

Lack of adequate genetic resistance to *R. solani* in the current cultivars of rapeseed and canola, and absence of cultural methods for suppressing the pathogen in the field, prompted redirection of research towards chemical control. Systematic screening of fungicides against *R. solani* damping-off and seedling root rot was initiated at the Agriculture and Agri-Food Canada Research Centre at Saskatoon (lat. 52°07' N, long. 106°38' W) in 1988.

Efficacy of seed treatments to control R. solani AG2-1-induced damping-off and seedling root rot in cultivars of Brassica spp.

In tests in a growth chamber, using soil-less mix infested with an isolate of *R. solani* AG2-1, benodanil [2-jodobenzanilide], carboxin [5,6-dihydro-2 methyl-1,4-oxathiine-3-carboxanilide], cyproconazole

[(2*RS*-3*RS*-2*RS*-3*SR*)-2-(4-chlorophenyl)-3-cyclopropyl-1(1*H*-1,2,4-triazol-1-yl)butan-2-ol], fenpropimorph [4-[3-[4-(1,1-dimethylethyl)phenyl]-2-methylpropyl]-2,6-(cis)-dimethylmorpholine], furmecyclohex [methyl *N*-cyclohexyl-2,5-dimethylfuran-3-carboxhydroxamate], iprodione [3-(3,5-dichlorophenyl)-*N*-isopropyl-2,4-dioxoimidazolidine-1-carboxamide], pencycuron [N-(4-chlorophenyl)-methyl-*N*-cyclopentyl-*N'*-phenylurea], and tolclofos-methyl [*O*-2,6-dichloro-*p*-tolyl *O*, *O*-dimethyl phosphorothioate] applied as seed treatments, at several rates, each controlled pre-emergence damping-off in one cultivar each of *B. rapa*, *B. napus*, *B. juncea*, *B. nigra*, *B. carinata* and *B. oleracea* (Kataria *et al.* 1993). The level of control at the lower rates of fungicides, however, differed among the *Brassica* species. Against damping-off, benodanil, furmecyclohex, iprodione and tolclofos-methyl provided high levels of control for all *Brassica* species, carboxin, cyproconazole and fenpropimorph were less effective in *B. oleracea*, and cyproconazole gave poorer control in *B. rapa* than in the other five species. Against post-emergence seedling root rot, furmecyclohex provided good control in all six *Brassica* species, benodanil, iprodione and tolclofos-methyl were most effective in *B. napus* and *B. juncea*, and cyproconazole gave moderate control in *B. napus* and poor control in the other five species tested.

In another growth chamber tests, cyproconazole at 0.1 g a.i. kg⁻¹ seed, and each of the seven other fungicides at 3 g a.i. kg⁻¹ seed, provided almost complete control of pre-emergence damping-off in all four cultivars of each of *B. juncea*, *B. napus* and *B. rapa* with little or no difference in the level of control among cultivars or fungicides (Kataria *et al.* 1993). Against post-emergence seedling root rot, efficacy of all eight fungicides, however, varied among cultivars within species, and there was a significant cultivar x fungicide interaction with most treatments. Furmecyclohex, pencycuron, tolclofos-methyl and iprodione provided excellent control of root rot, benodanil and fenpropimorph provided moderate control, and carboxin and cyproconazole gave poor control in all cultivars of the three species.

Since the six *Brassica* species differed considerably in seed weight and the number of seeds per gram (Kataria and Verma 1990), the amount of fungicide applied per seed was also different for each species. Thus differences in the level of disease control in different *Brassica* species may be due partly to differences in the amount of fungicide applied per seed. In addition, since *R. solani* AG2-1 differs in its capacity to form functional infection cushions on different rape-seed and mustard species (Yang *et al.* 1992b), fungicides may provide greater disease control on a relatively resistant cultivar/species, where *R. solani* can only form a few and less well organised infection cushions (Hofman and Jongebloed 1988).

Control, especially of post-emergence root rot, depended largely on the type of fungicides used for treating seed. Cyproconazole almost completely controlled pre-emergence damping-off, but gave poor control of post-emergence root rot, possibly because it is translocated rapidly in the stem and foliage, and does not remain in the root region long enough to control root rot (Cotterill *et al.* 1989). Carboxin, though effective against pre-emergence damping-off, also gave poor control against root rot, perhaps because it is readily oxidised to non fungitoxic sulfoxide in plant tissues and loses its effectiveness a few weeks after application (Briggs *et al.* 1974; Lyr *et al.* 1974; Snel and Edgington 1970).

Furmecyclox provided excellent control of both pre-emergence damping-off and post-emergence root rot in all *Brassica* species, presumably owing to its metabolic stability in the root tissue and intrinsically strong activity against *R. solani* (Huppatz *et al.* 1983; Martin and Torres 1986). Tolclofos-methyl and iprodione also gave excellent control of both damping-off and root rot in different *Brassica* species and cultivars because they both possess a strong fungitoxic activity (Kataria *et al.* 1991 a,b), show little or no acropetal mobility, and can accumulate in sufficient amounts in and around the roots and hypocotyls (Lyr 1987). Although pencycuron is effective against damping-off and root rot caused by *R. solani* AG2-1 isolates in *Brassica*

crops, it is ineffective against AG4 isolates, and thus would be of limited benefit in western Canada, where the plants are infected by both AG2-1 and AG4 isolates (Kataria *et al.* 1991b).

Interaction of fungicide and insecticide treatments

Carboxin and iprodione, widely used to control *R. solani* in canola in western Canada, normally are applied to seeds in formulations with the insecticide gamma-HCH [$1\alpha,2\alpha,3\beta,4\alpha,5\alpha,6\beta$ -hexachlorocyclohexane]. Effectiveness of the insecticide against flea beetles (*Phyllotreta* spp. [Coleoptera: Chrysomelidae]), the principal target insect, decreases rapidly during the week following seedling emergence (Westcott 1985). Systemic granular formulations of carbofuran [2,3-dihydro-2,2-dimethylbenzofuran-7-yl methyl carbamate] or terbufos [5-tert-butylthio-methyl 0,0-diethyl phosphoro dithioate] are applied in the seed furrow to extend the period of protection.

In tests in a growth chamber, evaluation of four insecticides, cloethocarb [2-(2-chloro-1-methoxyethoxy)-phenyl methylcarbamate], gamma-HCH, carbofuran and terbufos, alone and in combination with five fungicides (carboxin, cyproconazole, furmecyclox, iprodione and tolclofos-methyl) against *R. solani* AG2-1 in canola cv. Westar (*B. napus*), Kataria and Verma (1993) found that cloethocarb and carbofuran alone increased the incidence of damping-off and, when used in combination with fungicides, lowered the level of control of both damping-off and root rot. Gamma-HCH and terbufos decreased the incidence of damping-off, and terbufos also reduced seedling root rot. Combined treatments with gamma-HCH and terbufos gave better control than either gamma-HCH or terbufos alone. Disease control by fungicides was enhanced by the use of either gamma-HCH or terbufos together with fungicide treatments.

Maximum disease control with insecticide-fungicide combinations was achieved when terbufos granules were added at the time of seeding with fungicide + gamma-HCH-treated seed. Adding terbufos granules to the treated seed 1-4 wk before seeding resulted in a considerable loss of efficacy of each of the

five fungicide + gamma-HCH seed treatments. Although the level of disease control by gamma-HCH and terbufos was considerably lower than the control by any one of the five tested fungicides, it is significant that these two insecticides used in canola for the control of flea beetles, also offer some control against *R. solani* AG2-1-induced damping-off and root rot.

Carbofuran and cloethocarb increased disease incidence and lowered the efficacy of fungicides. The disease-controlling potential of gamma-HCH was also annulled when used in combination with carbofuran. The observed antagonism of carbofuran and cloethocarb with terbufos and with fungicides was perhaps due to the increased disease severity induced by carbofuran and cloethocarb (Hofman and Bollen 1987; Ruppel and Hecker 1982). Therefore, though carbofuran and cloethocarb possess good systemic activity in the control of flea beetles in canola (Romanow and Askew 1984; Wise 1989), our results indicate that these insecticides are not suitable components in a fungicide-insecticide mixture for the control of *R. solani* in canola (Kataria and Verma 1993).

Gamma-HCH provides some control of damping-off in canola because its aqueous solution and vapours are fungistatic to *R. solani* (Richardson and Miller 1960). Hence, depending on the amount of insecticide applied, gamma-HCH can affect fungal growth in soil at an appreciable distance from treated seed. Effectiveness of a mixture of gamma-HCH and seed dressing fungicides in controlling damping-off pathogens including *R. solani* has also been reported in peas (Richardson 1960).

Terbufos alone or in combination with fungicides, provided substantial control against damping-off and root rot in canola (Kataria and Verma 1993). Its activity, however, decreases after seedling emergence, presumably due to its absorption by organic matter in the soilless mix, rapid translocation to the foliage, or biological oxidation to less toxic compounds (Chapman and Harris 1980). Of the granular insecticides recommended in western Canada for flea beetle control in canola, only terbufos improved disease con-

trol against *R. solani* and interacted additively with fungicides and gamma-HCH. Accordingly, the maximum disease control was achieved when a fungicide was used in combination with both gamma-HCH and terbufos, and when terbufos granules were applied at the time of seeding the pots. Adding terbufos granules to the treated seed 1-4 wk before seeding reduced efficacy of each of the five fungicide + gamma-HCH seed treatments tested. Prior storage of canola seeds with terbufos granules impairs seed germination, retards seedling emergence, and predisposes the seeds and young seedlings to attack by microorganisms in soil (Askew and Romanow 1983; McKenzie *et al.* 1987). These factors could have rendered the seedlings more vulnerable to damping-off and root rot caused by *R. solani* in this study.

The studies of Kataria and Verma (1993) were conducted in a growth chamber and with soilless potting mix, so it is difficult to extrapolate the results to field conditions. Nevertheless, the results underscored the potential to improve control of canola damping-off and seedling root rot by developing combinations of insecticide and fungicide. Field studies are required to verify and quantify the beneficial effects of combining fungicide and insecticide so that the usefulness of their interactions can be predicted and utilized for efficient control of disease and insect pests.

In western Canada, seed treatments with commercial formulations such as carbothiin 4.5% + thiram 9% + lindane 67.5%, iprodione 16.7% + lindane 50%, and thiabendazole 1.6% + thiram 4.8% + lindane 40% are effective against damping-off and seedling root rot, but do not provide long-term control against the mature plant root rot and brown girdling root rot. Slow release fungicide granules might provide long-term disease control against root rot.

Biological control

Few studies have investigated the use of biocontrol agents to control damping-off and root rot in rapeseed/canola. In *B. napus* fields in Pakistan (Iqbal *et al.* 1977) observed that vesicular arbuscular-mycorrhizal plants suppressed root infection by *R. solani*, and Akhtar (1969)

obtained substantial control of damping-off in mustard plants grown in the soil amended with *Trichoderma harzianum*. Harman *et al.* (1980) also obtained substantial control of damping-off in radish plants in the soil amended with *T. hamatum*. Gugel *et al.* (1986) found that an indigenous population of non-pathogenic binucleate *Rhizoctonia* suppressed damping-off and root rot of canola caused by *R. solani* AG2-1 and AG4 in growth chamber and field tests. Dahiya and Woods (1987) isolated a strain of *Pseudomonas fluorescens* that produced three antibiotics (pyocyanin, pyrrolnitrin and phenazine carboxamide) inhibitory to *R. solani*; treating canola seed with *P. fluorescens* controlled damping-off in infested peat-mix soil. Reddy *et al.* (1994) evaluated seven bacterial strains (five of *P. fluorescens*; one of *Corynebacterium* group; and one of *Enterobacter agglomerans*) as canola seed treatments in field tests and identified two strains of *P. fluorescens* (63-49 and U-14) that were effective and consistent in suppressing *R. solani* irrespective of the field or year tested when compared to the non-bacterization controls.

REFERENCES

- Acharya, S.N., P.R. Verma, J. Dueck, and R.K. Downey. 1984. Screening rapeseed/canola for resistance to damping-off and seedling root rot caused by *Rhizoctonia solani*. Can. J. Plant Pathol. 6 : 325-328.
- Akhtar, C.M. 1969. Biological control of *Rhizoctonia solani* causing seed decay and damping-off of mustard. West Pakistan J. Agric. Res. 7 : 29-52.
- Askew, W.L., and W. Romanow. 1983. Effect of insecticide granules applied for control of flea beetles on germination of canola. Pestic. Res. Rep., Expert Committee on Pesticide Use in Agriculture, Ottawa, Canada. p. 82.
- Bateman, D.F. 1964. An induced mechanism of tissue resistance to polygalacturonase in *Rhizoctonia*-infected hypocotyls of bean. Phytopathology 54 : 438-445.
- Bateman, D.F. 1970. Pathogenicity and disease. Pages 161-171 in J.R. Parmeter Jr. (ed.), *Rhizoctonia solani*, biology and pathology. Univ. of California Press, Berkeley, U.S.A.
- Benhamou, N., K. Broglie, I. Chet, and R. Broglie. 1993. Cytology of infection of 35S-bean chitinase transgenic canola plants by *Rhizoctonia solani*: cytochemical aspects of chitin breakdown *in vivo*. Plant J. 4 : 295-305.
- Benson, D.M. 1973. Epidemiology of *Rhizoctonia solani* damping-off in radish. Phytopathology 63 : 1214 (Abstract).
- Benson, D.M., and R. Baker. 1974. Epidemiology of *Rhizoctonia solani* pre-emergence damping-off of radish: inoculum potential and disease potential interaction. Phytopathology 64 : 957-962.
- Berkenkamp, B. 1972. Diseases of rapeseed in central and northern Alberta in 1971. Can. Plant Dis. Surv. 52 : 62-63.
- Berkenkamp, B., and K. Degenhardt. 1974. Diseases of rapeseed in central and northern Alberta in 1972. Can. Plant Dis. Surv. 54 : 35-36.
- Berkenkamp, B., and H. Vaartnou. 1972. Fungi associated with rape root rot in Alberta. Can. J. Plant Sci. 52 : 973-976.
- Briggs, D.E., R.H. Waring, and A.M. Hackett. 1974. The metabolism of carboxin in growing barley. Pestic. Sci. 5 : 599-607.
- Broglie, K., I. Chet, M. Holliday, R. Cressman, P. Biddle, S. Knowlton, C.J. Mauvias, and R. Broglie. 1991. Transgenic plants with enhanced resistance to the fungal pathogen, *Rhizoctonia solani*. Science (Washington DC) 254 : 1194-1197.
- Calman, A.I., and J.P. Tewari. 1987. Involvement of *Pythium* spp. in the pre-emergence phase of canola seedling blight. Can. J. Plant Pathol. 9 : 274 (Abstract).
- Calman, A.I., J.P. Tewari, and M. Mugala. 1986. *Fusarium avenaceum* as one of the causal agents of seedling blight of canola in Alberta. Plant Dis. 70 : 694.
- Chapman, R.A., and C.R. Harris. 1980. Insecticidal activity and persistence of terbufos, terbufos sulfoxide and terbufos sulfone in soil. J. Econ. Entomol. 73 : 536-543.
- Chauhan, L.S., and H.K. Saxena. 1974. A new *Rhizoctonia* leaf blight of rapeseed and mustard. Indian J. Farm Sci. 2 : 98-99.
- Conn, K.L., J.P. Tewari, and J.S. Dahiya. 1987. Elicitation of phytoalexins in the roots of canola and *Camelina sativa* seedlings in response to *Rhizoctonia solani*. Proc. 8th Annu. Meet. Plant Pathol. Soc. Alberta, Lacombe, Alberta, 28-30 October, 1987. p. 18 (Abstract).
- Conn, K.L., L.M. Browne, J.P. Tewari, and W.A. Ayer. 1994. Resistance to *Rhizoctonia solani* and presence of antimicrobial compounds in *Camelina sativa* roots. J. Plant Biochem. Biotech. 3 : 125-130.

- Cotterill, P.J., D.J. Ballinger, and J.F. Kollmorgen. 1989. Use of three screening techniques for the evaluation of fungicides to control *Rhizoctonia* root rot of wheat. *Ann. Appl. Biol.* 115 : 229-235.
- Dahiya, J.S., and D.L. Woods. 1987. Control of *Rhizoctonia solani*, causal agent of brown girdling root rot of canola/rapeseed by *Pseudomonas fluorescens*. *Can. J. Plant Pathol.* 9 : 275-276.
- Dodman, R.L., K.R. Barker, and J.C. Walker. 1968. A detailed study of the different modes of penetration by *Rhizoctonia solani*. *Phytopathology* 58 : 1271-1276.
- Ellis, P.J. 1983. Diseases of rapeseed in the Peace River region of Alberta. M.P.M. Thesis. Simon Fraser University, Burnaby, B.C., Canada. 89 pp.
- Flentje, N.T., R.L. Dodman, and A. Kerr. 1963. The mechanisms of host penetration by *Thanatephorus cucumeris*. *Aust. J. Biol. Sci.* 16 : 784-799.
- Gugel, R.K., P.R. Verma, and D.A. Kaminski. 1986. Protection of canola seedlings from *Rhizoctonia solani* infection by a nonpathogenic, binucleate *Rhizoctonia*-like isolate. *Can. J. Plant Pathol.* 8 : 349 (Abstract).
- Gugel, R.K., S.M. Yitbarek, P.R. Verma, R.A.A. Morrall, and R.S. Sadasivaiah. 1987. Etiology of the rhizoctonia root rot complex of canola in the Peace River region of Alberta. *Can. J. Plant Pathol.* 9 : 119-128.
- Gupta, D.K. 1985. Root and collar rot of mustard in sikkim. *Indian J. Mycol. Plant Pathol.* 15 : 225.
- Harman, G.E., I. Chet, and R. Baker. 1980. *Trichoderma hamatum* effects on seed and seedling disease induced in radish and pea by *Pythium* spp. or *Rhizoctonia solani*. *Phytopathology* 70 : 1167-1172.
- Harrison, L.M. 1987. Evaluation of *Brassica* accessions for resistance to *Rhizoctonia solani* and the effects of seeding date, soil temperature and moisture on disease development. M.Sc. Thesis, University of Manitoba, Winnipeg, Canada. 109 pp.
- Henis, Y., A. Ghaffar, and R. Baker. 1978. Integrated control of *Rhizoctonia solani* damping-off of radish : effect of successive plantings, PCNB, and *Trichoderma harzianum* on pathogen and disease. *Phytopathology* 68 : 900-907.
- Hofman, T.W., and G.J. Bollen. 1987. Effects of granular nematicides on growth and microbial antagonism to *Rhizoctonia solani*. *Neth. J. Plant Pathol.* 93 : 201-214.
- Hofman, T.W., and P.H.J. Jongebloed. 1988. Infection process of *Rhizoctonia solani* on *Solanum tuberosum* and effects of granular nematicides. *Neth. J. Plant Pathol.* 94 : 243-252.
- Homma, Y., and M. Ishii. 1984. [Anastomosis groups of *Rhizoctonia solani* Kühn responsible for various symptoms of the root rot of Japanese radish.] (in Japanese) *Bull. Shikoku Agric. Exp. Stn. No.* 42 : 1-11.
- Huber, D.M., E.P. Christmas, L.J. Herr, T.S. McCay-Buis, and R. Baird. 1992. *Rhizoctonia* crown rot of canola in Indiana. *Plant Dis.* 76 : 1251-1253.
- Huppertz, J.L., J.N. Phillips, and B. Witzens. 1983. Laboratory and glasshouse studies of the activity of carboxamide derivatives against *Rhizoctonia solani* in cotton. *Plant Dis.* 67 : 45-47.
- Hwang, S.F., T.A. Swanson, and I.R. Evans. 1986. Characterization of *Rhizoctonia solani* isolates from canola in west central Alberta. *Plant Dis.* 70 : 681-683.
- Iqbal, S.H., K.S. Kureshi, and J.S. Ahmad. 1977. Influence of vesicular arbuscular mycorrhiza on damping-off caused by *Rhizoctonia solani* in *Brassica napus*. *Biologia* 23 : 197-208.
- Johnson, A.W., A.M. Golden, D.L. Auld, and D.R. Sumner. 1992. Effects of rapeseed and vetch as green manure crops and fallow on nematodes and soil-borne pathogens. *J. Nematol.* 24 : 117-126.
- Kaminski, D.A., and P.R. Verma. 1985. Cultural characteristics, virulence, and *in vitro* temperature effect on mycelial growth of *Rhizoctonia* isolates from rapeseed. *Can. J. Plant Pathol.* 7 : 256-261.
- Kataria, H.R., and P.R. Verma. 1990. Efficacy of fungicidal seed treatments against pre-emergence damping-off and post-emergence seedling root rot of growth chamber grown canola caused by *Rhizoctonia solani* AG2-1 and AG4. *Can. J. Plant Pathol.* 12 : 409-416.
- Kataria, H.R., and P.R. Verma. 1992. *Rhizoctonia solani* damping-off in oilseed rape and canola. *Crop Prot.* 11 : 8-13.
- Kataria, H.R., and P.R. Verma. 1993. Interactions of fungicide and insecticide combinations against *Rhizoctonia* damping-off and root rot in canola. *Ann. Appl. Biol.* 123 : 233-246.
- Kataria, H.R., U. Hugelshofer, and U. Gisi. 1991a. Sensitivity of *Rhizoctonia* species to different fungicides. *Plant Pathol.* 40 : 203-211.
- Kataria, H.R., P.R. Verma, and U. Gisi. 1991b. Variability in the sensitivity of *Rhizoctonia solani* anastomosis groups to fungicides. *J. Phytopathol.* 133 : 121-133.
- Kataria, H.R., P.R. Verma, and G. Rakow. 1993. Fungicidal control of damping-off and seedling root rot in *Brassica* species caused by *Rhizoctonia solani* in the growth chamber. *Ann. Appl. Biol.* 123 : 247-256.

- Keyworth, W.G., and J.M. Dow. 1960.** The control of damping-off and wirestem of *Brassica* seedlings caused by *Rhizoctonia solani* Kühn. Annu. Rep. National Vegetable Res. Stn., Wellesbourne, Warwick, U.K. pp. 51-54.
- Lyr, H. 1987.** Modern selective fungicides. Properties, applications and mechanisms of action. Wiley, New York. 383 pp.
- Lyr, H., G. Ritter, and L. Banasiak. 1974.** Detoxification of carboxin. Z. Allg. Mikrobiol. 14 : 313-320.
- Mahmood, T., and S.H. Iqbal. 1982.** Influence of soil moisture contents on VA mycorrhizal and pathogenic infection by *Rhizoctonia solani* in *Brassica napus*. Pak. J. Agric. Res. 3 : 45-49.
- Martin, B.C., and H. Torres. 1986.** Fungicides for the control of *Rhizoctonia solani* damping-off in seedlings derived from true potato seed. Fitopatologia 21 : 74-80.
- Massaquoi, R.C. and M.C. Rush. 1987.** Relationship of quantity of epicuticular wax to resistance of rice to sheath blight. Phytopathology 77 : 17-23.
- McKenzie, D.L., P.R. Verma, and R.K. Gugel. 1987.** Field experiments on the effects of combinations of 2 seed dressing fungicides and 2 granular insecticides on emergence of canola, 1987. Pestic. Res. Rep., Expert Committee on Pesticide Use in Agriculture, Ottawa, Canada. pp. 213-214.
- Mohapatra, D., and Y.P.S. Bajaj. 1987.** Inter-specific hybridization in *Brassica juncea* x *Brassica hirta* using embryo rescue. Euphytica 36 : 321-326.
- Petrie, G.A. 1973.** Diseases of *Brassica* species in Saskatchewan. 1970-72. III, Stem and root rots. Can. Plant Dis. Surv. 53 : 88-92.
- Petrie, G.A., and T.C. Vanterpool. 1966.** Diseases of rape, mustard and cruciferous weeds in the Prairie provinces. Can. Plant Dis. Surv. 46 : 117-120.
- Petrie, G.A., and T.C. Vanterpool. 1970.** Diseases of rape and other crucifers in Saskatchewan in 1969. Can. Plant Dis. Surv. 50 : 106-107.
- Reddy, M.S., D.C. Covert, K.A. Craig, R.J. Rennie, D. McKenzie, P.R. Verma, and K. Mortensen. 1994.** Suppression of pre-emergence damping-off of canola, caused by *Rhizoctonia solani*, by rhizobacteria. Pages 80-82 in M.H. Ryder, P.M. Stephens, and G.D. Bowen (eds.), Improving plant productivity with rhizosphere bacteria. Proc. 3rd Int. Workshop on plant growth promoting rhizobacteria, Adelaide, South Australia, 7-11 March, 1994.
- Richardson, L.T. 1960.** Effect of insecticide-fungicide combinations on emergence of peas and growth of damping-off fungi. Plant Dis. Rep. 44 : 104-108.
- Richardson, L.T., and D.M. Miller. 1960.** Fungitoxicity of chlorinated hydrocarbon insecticides in relation to water solubility and vapour pressure. Can. J. Bot. 38 : 163-175.
- Rimmer, S.R., and R.G. Platford. 1982.** Manitoba rapeseed disease survey 1978-1980. Can. Plant Dis. Surv. 62 : 45-49.
- Ripley, V.L., and P.G. Arnison. 1990.** Hybridization of *Sinapis alba* L. and *Brassica napus* L. via embryo rescue. Plant Breeding 104 : 26-33.
- Romanow, W., and W.L. Askew. 1984.** Insecticide trials against flea beetles on canola. Pestic. Res. Rep., Expert Committee on Pesticide Use in Agriculture, Ottawa, Canada. pp. 101-102.
- Ruppel, E.G., and R.J. Hecker. 1982.** Increased severity of *Rhizoctonia* root rot in sugar beet treated with systemic insecticides. Crop Prot. 1 : 75-81.
- Sippell, D.W., J.G.N. Davidson, and M. Cox. 1985a.** Factors affecting severity of root rot of canola in the Peace River region. Proc. 6th Annu. Meet. Plant Pathol. Soc. Alberta, Lethbridge, Alberta. 7-8 November 1985 p. 34. (Abstract).
- Sippell, D.W., J.G.N. Davidson, and R.S. Sadasivaiah. 1985b.** *Rhizoctonia* root rot of rapeseed in the Peace River region of Alberta. Can. J. Plant Pathol. 7 : 184-186.
- Snel, M., and L.V. Edgington. 1970.** Uptake, translocation and decomposition of systemic oxathiin fungicides in bean. Phytopathology 60 : 1708-1716.
- Stockwell, V., and P. Hanchey. 1983.** The role of the cuticle thickness on infection in resistance of beans to *Rhizoctonia solani*. Phytopathology 73 : 1640-1642.
- Tahvonen, R., J. Hollo, A. Hannukkala, and A. Kurppa. 1984.** *Rhizoctonia solani* damping-off on spring turnip rape and spring rape (*Brassica* spp.) in Finland. J. Agric. Sci. Finl. 56 : 143-154.
- Teo, B.K., S.M. Yitbarek, P.R. Verma, and R.A.A. Morrall. 1988.** Influence of soil moisture, seeding date, and *Rhizoctonia solani* isolates (AG2-1 and AG4) on disease incidence and yield in canola. Can. J. Plant Pathol. 10 : 151-158.
- Tewari, J.P., K.L. Conn, and J.S. Dahiya. 1988.** Dynamic response of rapeseed and other crucifers to *Alternaria brassicae* and *Rhizoctonia solani*. Proc. 5th Int. Cong. Plant Pathol., Kyoto, Japan. 242 pp.
- Van Lelivelt, C.L., H.J. Furderiks, and N. Van Dijk. 1989.** Transfer of resistance to beet cyst nematode to Swede rape via interspecific crosses with related species. Plant Breed. Abstr. 59 : 59.
- Vanterpool, T.C. 1960.** Rape diseases in Saskatchewan in 1960. Can. Plant Dis. Surv. 40 : 60-61.

- Vanterpool, T.C. 1963.** Rape diseases in Saskatchewan in 1963. Can. Plant Dis. Surv. 43 : 212-214.
- Vanterpool, T.C. 1974.** *Pythium polymastum* pathogenic on oilseed rape and other crucifers. Can. J. Bot. 52 : 1205-1208.
- Verma, P.R., and H.R. Kataria. 1990.** Damping-off and root rot in rapeseed/canola caused by *Rhizoctonia solani* - an overview. Proc. 3rd Int. Cong. Plant Prot. in the Tropics, Kuala Lumpur, Malaysia 6 : 183-187.
- Vidhyasekaran, P. 1988.** Physiology of disease resistance in plants. Vol. 2. CRC Press, Boca Raton, Florida. 128 p.
- Weber, Z. 1987.** Susceptibility of rape to fungi causing root rot. Proc. 7th Int. Rapeseed Congr., Poznan, Poland. pp. 1193-1197.
- Westcott, N.D. 1985.** Gamma-HCH in rape seedlings grown from treated seeds. Pestic. Sci. 16 : 416-421.
- Williams, P.H., and J.C. Walker. 1966.** Inheritance of *Rhizoctonia* bottom rot resistance in cabbage. Phytopathology 56 : 367-368.
- Wise, I.L. 1989.** Flea beetle control in canola with seed coated and in-furrow granular insecticides. Pestic. Res. Rep., Expert Committee on Pesticide Use in Agriculture, Ottawa, Canada. pp. 50-51.
- Yang, J. 1989.** Studies on *Rhizoctonia solani* diseases of rapeseed : germplasm screening, histo-cytopathology, cuticle thickness and calcium content in relation to resistance. M.Sc. Thesis, University of Saskatchewan, Saskatoon, Canada. 141 pp.
- Yang, J., and P.R. Verma. 1992.** Screening genotypes for resistance to pre-emergence damping-off and post-emergence seedling root rot of oilseed rape and canola caused by *Rhizoctonia solani* AG2-1. Crop Prot. 11 : 443-448.
- Yang, J., P.R. Verma, and G.L. Lees. 1992a.** The role of cuticle and epidermal cell wall in resistance of rapeseed and mustard to *Rhizoctonia solani*. Plant Soil 142 : 315-321.
- Yang, J., P.R. Verma, and J.P. Tewari. 1992b.** Histopathology of resistant mustard and susceptible canola hypocotyls infected by *Rhizoctonia solani*. Mycol. Res. 96 : 171-179.
- Yang, J., J.P. Tewari, and P.R. Verma. 1993.** Calcium oxalate crystal formation in *Rhizoctonia solani* AG2-1 culture and infected crucifer tissue : relationship between host calcium and resistance. Mycol. Res. 97 : 1516-1522.
- Yang, J., P.D. Kharbanda, H. Wang, and D.W. McAndrew. 1996.** Characterization, virulence and genetic variation of *Rhizoctonia solani* AG9 in Alberta. Plant Dis. 80 : 513-518.
- Yitbarek, S.M. 1987.** Studies of *Rhizoctonia* diseases of rapeseed/canola in Saskatchewan. M.Sc. Thesis, University of Saskatchewan, Saskatoon, Canada. 114 pp.
- Yitbarek, S.M., P.R. Verma, and R.A.A. Morrall. 1987.** Anastomosis groups, pathogenicity, and specificity of *Rhizoctonia solani* isolates from seedling and adult rapeseed/canola plants and soils in Saskatchewan. Can. J. Plant Pathol. 9 : 6-13.
- Yitbarek, S.M., P.R. Verma, R.K. Gugel, and R.A.A. Morrall. 1988.** Effect of soil temperature and inoculum density on pre-emergence damping-off of canola caused by *Rhizoctonia solani*. Can. J. Plant Pathol. 10 : 93-98.